IN Brin, Mitchell F.; Donovan, Stephen
PA Allergan Sales, Inc, USA
SO U.S. Pat. Appl. Publ., 19 pp., Cont.-in-part of U.S. Ser. No. 631,221.
CODEN: USXXCO
DT Patent
LA English
FAN.CNT 3

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	US 2002094339	A1	20020718	US 2002-71826	20020208
	US 6139845	A	20001031	US 1999-454842	19991207
PRAI	US 1999-454842	A2	19991207		
	US 2000-631221	A2	20000802		

AB A method for treating a mammary gland disorder, including hyperplastic, hypertonic, cystic and/or neoplastic mammary gland tissue by local administration of a **botulinum** toxin to or to the vicinity of the afflicted breast tissue is described.

L26 ANSWER 5 OF 11 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC. on STN
AN 2001:562774 BIOSIS
DN PREV200100562774
TI Gangliosides confer sensitivity to **botulinum** neurotoxin A.
AU Bateman, K. E. (1); Keller, J. E. (1); Neale, E. A. (1)
CS (1) Lab. Dev. Neurobiol., Natl. Inst. of Child Health and Human Dev., NIH, Bethesda, MD USA
SO Society for Neuroscience Abstracts, (2001) Vol. 27, No. 2, pp. 1876. print.
Meeting Info.: 31st Annual Meeting of the Society for Neuroscience San Diego, California, USA November 10-15, 2001
ISSN: 0190-5295.
DT Conference
LA English
SL English
AB The clostridial neurotoxins (tetanus and **botulinum**) act presynaptically by cleaving proteins required for synaptic vesicle exocytosis. Specific high affinity neuronal surface receptor(s) for the clostridial neurotoxins have not been identified although these toxins bind with low affinity to polysialogangliosides. Tetanus toxin-induced blockade of neurotransmitter release is prevented in neuronal cultures maintained in the drug fumonisin B1, which prevents polysialoganglioside synthesis by inhibiting ceramide synthase. Addition of exogenous gangliosides to fumonisin-treated cultures restores tetanus toxin binding and its ability to block neurotransmitter release (Williamson et al., 1999, J Biol Chem 274:25173-25180). Thus, in the case of tetanus toxin, gangliosides are critical for the delivery of **toxin** to its intracellular **substrate**. In this study, we show that fumonisin B1 (20muM for three weeks) confers significant protection to mouse spinal cord cell cultures against **botulinum** neurotoxin A-induced inhibition of neurotransmitter release and proteolysis of **SNAP-25**. Furthermore, the addition of gangliosides to ganglioside-depleted cultures restores the ability of **botulinum** neurotoxin A to block potassium-evoked glycine release. The gangliosides GT1b and GQ1b are nearly equivalent and GD1b is somewhat less effective in restoring toxin action. These results suggest that gangliosides play an important role in mediating the binding and delivery of **botulinum** neurotoxin A.

L26 ANSWER 6 OF 11 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC. on STN
DUPLICATE 2
AN 2001:184024 BIOSIS
DN PREV200100184024
TI Cleavage of **SNAP-25** by **botulinum** toxin type A requires receptor-mediated endocytosis, pH-dependent translocation, and